Table 1 Release of endogenous amino acids from the cat spinal cord *in vivo*

Amino acid	*Release following:*	
	pCMS 10^{-4} M (% spontaneous) A	*pCMS 10^{-4} M + stimulation (% pCMS 10^{-4} M release) B*
Leucine	186 ± 26 (10)†	137 ± 11 (4)‡
Alanine	233 ± 57 (7)	229 ± 67 (4)‡
Aspartate	179 ± 30 (8)*	264 ± 102 (3)
GABA	387 ± 98 (10)*	163 ± 11 (4)§
Glutamate	197 ± 25 (8)†	186 ± 13 (3)‡
Glutamine	100 ± 6 (10)	Not calculated
Glycine	850 ± 142 (10)†	194 ± 41 (4)‡
Lysine	72 ± 8 (5)*	130 ± 21 (3)
Proline	256 ± 60 (6)*	169 ± 29 (4)

Spontaneous release of amino acids was determined in the absence of pCMS. The ^3H radioactivity in each dansyl derivative was corrected for recovery by means of a [^{14}C] leucine internal standard and values were normalized with respect to the spontaneous efflux of endogenous leucine.

A: Release after 60 min perfusion with pCMS 10^{-4} M in artificial CSF. Values are per cent of spontaneous release.

B: Maximum release following stimulation in the presence of pCMS 10^{-4} M. Values are per cent of pCMS 10^{-4}M release.

All values are mean ± s.e. mean (number of observations in parentheses). Significance levels (2-tail paired *t*-test): $P<0.05$* or 0.01† compared with spontaneous release. $P<0.05$‡ or 0.01§ compared with release following pCMS 10^{-4} M. For all other comparisons $P>0.1$.

of leucine (not a neurotransmitter candidate) indicates that caution is necessary in this interpretation.

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The effect of cortisol on α_1-macroglobulin and α_2-acute phase globulin in the arthritic rat

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Recent work has shown that plasma antiproteases may be anti-inflammatory since they inhibit a number of proteases that are involved in inflammation (Barrett & Starkey, 1973; Hercz, 1974). The two most abundant human plasma antiproteases are α_1-antitrypsin and α_2-macroglobulin. In rat plasma the corresponding pair of antiproteases are α_1-antitrypsin and α_1-macroglobulin. A third antiprotease, α_2-acute phase globulin, is produced as a result of inflammation. Both α_1-macroglobulin and α_2-acute phase globulin are similar in properties to human α_2-macroglobulin and can be assayed enzymatically by

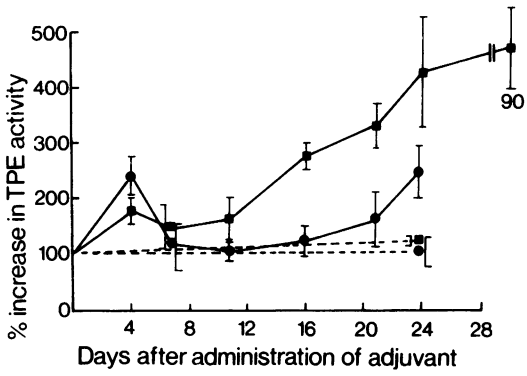

Figure 1 Trypsin-protein-esterase (TPE) values of the plasma of arthritic rats ■——■ and arthritic rats treated with cortisol ●——●. Dotted lines represent non-arthritic control animals. Each result represents the mean of five animals and vertical lines indicate s.e. mean.

measuring their trypsin-protein esterase (TPE) activity (Ganrot, 1973).

In this communication we report on the action of cortisol on the plasma levels of α_1-macroglobulin and α_2-acute phase protein in the normal and adjuvant arthritic rat.

Adjuvant arthritis was induced in male, Wistar strain rats (180–200 g body weight) (Parrott & Lewis, 1974). Some normal and some arthritic rats were injected daily, subcutaneously, with cortisol acetate in saline (5 mg/kg body weight) over the experimental period. The controls were injected with saline. α_1-macroglobulin and α_2-acute phase globulin were assayed simultaneously by measuring the TPE activity of plasma.

The results show that TPE plasma levels were elevated by inflammation but that cortisol treatment depressed these levels towards that of the non-arthritic controls. Cortisol had no significant effect on non-Arthritic rat plasma TPE levels.

Cortisol stimulates α_1-antitrypsin levels in the blood of both normal and arthritic rats (Parrott & Lewis, 1976) but clearly it has no effect on α_1-macroglobulin or α_2-acute phase globulin levels apart from depressing acute phase antiproteases through its anti-inflammatory action.

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Potentiation of anaphylactic bronchoconstriction by non-steroidal anti-inflammatory agents

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Non-steroidal anti-inflammatory agents have been reported to enhance mediator release from both human and guinea-pig sensitized lung challenged *in vitro* (Walker, 1972; Engineer, Piper & Sirois, 1976). We have extended these observations to the *in vivo* situation and report here the effect of non-steroidal anti-inflammatory agents on anaphylactic bronchoconstriction in the guinea-pig.

Guinea-pigs were sensitized either passively or actively. Passive sensitization was by intravenous injection of 0.5 ml of a 1/50 dilution of serum prepared according to the method of Davies & Johnston (1971) and were challenged 24 h later. Active sensitization was by the intraperitoneal and subcutaneous injection of ovalbumen (100 mg/kg) with antigen challenge three weeks later. Anaphylactic bronchoconstriction was measured according to the method of Collier & James (1967). Inhibitory compounds and antigen (ovalbumen) were administered intravenously, the compounds being given 5 min before antigen.

Sodium meclofenamate (1 mg/kg) significantly potentiated anaphylactic bronchoconstriction in passively sensitized guinea-pigs when submaximal doses of antigen (0.12–0.60 mg/kg) were used for challenge. When animals were challenged with a maximal dose of antigen (15 mg/kg), sodium meclofenamate had no significant effect. In animals challenged with a maximal dose of antigen in which the histamine-induced component of bronchoconstriction had been suppressed by the administration of mepyramine (2 mg/kg), sodium meclofenamate potentiated the reaction, returning the bronchoconstriction to the level of that seen in control animals.